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| NEWS 1 | | Web Page for STN Seminar Schedule - N. America |
| NEWS 2 | DEC 01 | ChemPort single article sales feature unavailable |
| NEWS 3 | JUN 01 | CAS REGISTRY Source of Registration (SR) searching enhanced on STN |
| NEWS 4 | JUN 26 | NUTRACEUT and PHARMAML no longer updated |
| NEWS 5 | JUN 29 | IMSCOPROFILE now reloaded monthly |
| NEWS 6 | JUN 29 | EPFULL adds Simultaneous Left and Right Truncation (SLART) to AB, MCLM, and TI fields |
| NEWS 7 | JUL 09 | PATDPAFULL adds Simultaneous Left and Right Truncation (SLART) to AB, CLM, MCLM, and TI fields |
| NEWS 8 | JUL 14 | USGENE enhances coverage of patent sequence location (PSL) data |
| NEWS 9 | JUL 27 | CA/CAplus enhanced with new citing references |
| NEWS 10 | JUL 16 | GBFULL adds patent backfile data to 1855 |
| NEWS 11 | JUL 21 | USGENE adds bibliographic and sequence information |
| NEWS 12 | JUL 28 | EPFULL adds first-page images and applicant-cited references |
| NEWS 13 | JUL 28 | INPADOCDB and INPAFAMDB add Russian legal status data |
| NEWS 14 | AUG 10 | Time limit for inactive STN sessions doubles to 40 minutes |
| NEWS 15 | AUG 18 | COMPENDEX indexing changed for the Corporate Source (CS) field |
| NEWS 16 | AUG 24 | ENCOMPLIT/ENCOMPLIT2 reloaded and enhanced |
| NEWS 17 | AUG 24 | CA/CAplus enhanced with legal status information for U.S. patents |
| NEWS 18 | SEP 09 | 50 Millionth Unique Chemical Substance Recorded in CAS REGISTRY |
| NEWS 19 | SEP 11 | WPIDS, WPINDEX, and WPIX now include Japanese FTERM thesaurus |

NEWS EXPRESS MAY 26 09 CURRENT WINDOWS VERSION IS V8.4,
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=> s (casein kinase I gamma) or (casein kinase 1 gamma) or ck1-g or ck1g or
ck1-gamma or ck1gamma or ck1-  or ck1  or CSNK1G
L1         1813 (CASEIN KINASE I GAMMA) OR (CASEIN KINASE 1 GAMMA) OR CK1-G OR
                CK1G OR CK1-GAMMA OR CK1GAMMA OR CK1- OR CK1 OR CSNK1G

=> S p21 or cip1 or WAF1 or (Cyclin-dependent kinase inhibitor 1A) or CDKN1A
L2       136615 P21 OR CIP1 OR WAF1 OR (CYCLIN-DEPENDENT KINASE INHIBITOR 1A)
                OR CDKN1A

=> s l1 (P) l2
L3          17 L1 (P) L2

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PROCESSING COMPLETED FOR L3
L4           12 DUPLICATE REMOVE L3 (5 DUPLICATES REMOVED)

=> d l4 1-12

L4   ANSWER 1 OF 12  BIOSIS  COPYRIGHT (c) 2009 The Thomson Corporation  on STN
AN   2009:136475  BIOSIS
DN   PREV200900136475
TI   Dietary Carbohydrate Source Alters Gene Expression Profile of Intestinal
     Epithelium in Mice.
AU   Wang, Bing [Reprint Author]; Bobe, Gerd; LaPres, John J.; Bourquin, Leslie
     D.
CS   Michigan State Univ, Dept Food Sci and Human Nutr, E Lansing, MI 48824 USA
     bourqui1@msu.edu
SO   Nutrition and Cancer, (2009) Vol. 61, No. 1, pp. 146-155.
     CODEN: NUCADQ. ISSN: 0163-5581.
DT   Article
LA   English
ED   Entered STN: 18 Feb 2009
     Last Updated on STN: 18 Feb 2009
```

L4 ANSWER 2 OF 12 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
AN 2009176133 EMBASE
TI Advances in the development of kinase inhibitor therapeutics for alzheimer's disease.
AU Savage, Mary J.
CS Merck and Company, West Point, PA 19486. mary_savage@merck.com
AU Gingrich, Diane E.
CS Cephalon, Inc., West Chester, PA 19380.
AU Savage, M. J., Dr. (correspondence)
CS Merck and Company, West Point, PA 19486. mary_savage@merck.com
SO Drug Development Research, (March 2009) Vol. 70, No. 2, pp. 125-144. Refs: 221
ISSN: 0272-4391; E-ISSN: 1098-2299 CODEN: DDREDK
PB Wiley-Liss Inc., 111 River Street, Hoboken, NJ 07030-5774, United States.
CY United States
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LA English
SL English
ED Entered STN: 5 May 2009
Last Updated on STN: 5 May 2009

L4 ANSWER 3 OF 12 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 2009:277956 BIOSIS
DN PREV200900277956
TI beta-tting on p63 as a Metastatic Suppressor.
AU Clohessy, John G.; Pandolfi, Pier Paolo [Reprint Author]
CS Harvard Univ, Sch Med, Beth Israel Deaconess Canc Ctr, Canc Gene Program, Boston, MA 02215 USA
ppandolf@bidmc.harvard.edu
SO Cell, (APR 2 2009) Vol. 137, No. 1, pp. 28-31.
CODEN: CELLB5. ISSN: 0092-8674.
DT Article
Editorial
LA English
ED Entered STN: 30 Apr 2009
Last Updated on STN: 30 Apr 2009

L4 ANSWER 4 OF 12 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 2006:263861 BIOSIS
DN PREV200600263250
TI Hematopoietic stem cell exhaustion impacted by p18(INK4C) and p21(Cip1/Waf1) in opposite manners.
AU Yu, Hui; Yuan, Youzhong; Shen, Hongmei; Cheng, Tao [Reprint Author]
CS Hillman Canc Ctr Res Pavil, 5117 Ctr Ave,Room 2-42E, Pittsburgh, PA 15213 USA
chengt@upmc.edu
SO Blood, (FEB 1 2006) Vol. 107, No. 3, pp. 1200-1206.
CODEN: BLOOAW. ISSN: 0006-4971.
DT Article
LA English
ED Entered STN: 10 May 2006
Last Updated on STN: 10 May 2006

L4 ANSWER 5 OF 12 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 2007:260865 BIOSIS
DN PREV200700270932

TI Cultured human mammary epithelial cell senescence barriers and hTERT expression.
AU Stampfer, Martha R. [Reprint Author]; Tlsty, Thea; Bazarov, Alex; Yaswen, Paul; Garbe, James
CS Lawrence Berkeley Natl Lab, Berkeley, CA USA
SO Proceedings of the American Association for Cancer Research Annual Meeting, (APR 2005) Vol. 46, pp. 666-667.
Meeting Info.: 96th Annual Meeting of the American-Association-for-Cancer-Research. Anaheim, CA, USA. April 16 -20, 2005. Amer Assoc Canc Res.
ISSN: 0197-016X.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 25 Apr 2007
Last Updated on STN: 11 Jul 2007

L4 ANSWER 6 OF 12 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2004:143261 CAPLUS
DN 140:176313
TI casein kinase I gamma-1 isoforms (CSNK1G1s) as modifiers of the p21 pathway and uses thereof in diagnosis, therapy and drug screening
IN Francis-Lang, Helen; Friedman, Lori; Kidd, Thomas; Roche, Siobhan; Zhang, Haiguang
PA Exelixis, Inc., USA
SO PCT Int. Appl., 69 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 8

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|---|
| PI | WO 2004015071 | A2 | 20040219 | WO 2003-US24551 | 20030806 |
| | WO 2004015071 | A3 | 20040812 | | |
| | W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW | | | | |
| | RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| | CA 2494236 | A1 | 20040219 | CA 2003-2494236 | 20030806 |
| | AU 2003263995 | A1 | 20040225 | AU 2003-263995 | 20030806 |
| | EP 1534852 | A2 | 20050601 | EP 2003-784937 | 20030806 |
| | R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK | | | | |
| | JP 2005534334 | T | 20051117 | JP 2004-527773 | 20030806 |
| | US 20050251870 | A1 | 20051110 | US 2005-523588 | 20050204 |
| PRAI | US 2002-401739P | P | 20020807 | | |
| | WO 2003-US24551 | W | 20030806 | | |

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OSC.G 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

L4 ANSWER 7 OF 12 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 2002:112722 BIOSIS
DN PREV200200112722
TI Differential cyclin-dependent kinase inhibitor CKI and INK4A expression in

canine breast cancer.
AU Lynn, Kristie A. [Reprint author]; DeInnocentes, Patricia [Reprint author]; Gwin, William R. [Reprint author]; Bird, R. Curtis [Reprint author]
CS Pathobiology, Auburn University, Auburn, AL, USA
SO Molecular Biology of the Cell, (Nov, 2001) Vol. 12, No. Supplement, pp. 11a. print.
Meeting Info.: 41st Annual Meeting of the American Society for Cell Biology. Washington DC, USA. December 08-12, 2001. American Society for Cell Biology.
CODEN: MBCEEV. ISSN: 1059-1524.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 30 Jan 2002
Last Updated on STN: 26 Feb 2002

L4 ANSWER 8 OF 12 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN DUPLICATE 1
AN 1999268046 EMBASE
TI Angiotensin II stimulates serine phosphorylation of the adaptor protein Nck: Physical association with the serine/threonine kinases Pak1 and casein kinase I.
AU Voisin, Laure; Meloche, Sylvain (correspondence)
CS Centre de Recherche, Ctr. Hosp. de l'Univ. de Montreal, University of Montreal, 3850 St. Urbain, Montreal, Que. H2W 1T8, Canada. meloches@ere.umontreal.ca
AU Larose, Louise
CS Department of Experimental Medicine, McGill University, Montreal, Que. H3A 2B2, Canada.
AU Meloche, Sylvain (correspondence)
CS Centre de Recherche, Centre hospitalier Univ. de Montreal, Campus Hotel-Dieu, 3850 St. Urbain, Montreal, Que. H2W 1T8, Canada. meloches@ere.umontreal.ca
SO Biochemical Journal, (1 Jul 1999) Vol. 341, No. 1, pp. 217-223.
Refs: 44
ISSN: 0264-6021 CODEN: BIJOAK
CY United Kingdom
DT Journal; Article
FS 029 Clinical and Experimental Biochemistry
LA English
SL English
ED Entered STN: 12 Aug 1999
Last Updated on STN: 12 Aug 1999

L4 ANSWER 9 OF 12 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 1999:10466 BIOSIS
DN PREV199900010466
TI Lovastatin mediated G1 arrest in normal and tumor breast cells is through inhibition of CDK2 activity and redistribution of p21 and p27, independent of p53.
AU Rao, Sharmila; Lowe, Michael; Herliczek, Thaddeus W.; Keyomarsi, Khandan [Reprint author]
CS Lab. Diagnostic Oncol., Div. Molecular Med., Wadsworth Cent., Albany, NY 12201-0509, USA
SO Oncogene, (Nov. 5, 1998) Vol. 17, No. 18, pp. 2393-2402. print.
CODEN: ONCNES. ISSN: 0950-9232.
DT Article
LA English
ED Entered STN: 11 Jan 1999
Last Updated on STN: 11 Jan 1999

L4 ANSWER 10 OF 12 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 1998:326689 BIOSIS
DN PREV199800326689
TI The cyclin kinase inhibitor p21WAF1,CIP1 is increased in experimental diabetic nephropathy: Potential role in glomerular hypertrophy.
AU Kuan, Chia-Jen; Al-Douahji, Mouhannad; Shankland, Stuart J. [Reprint author]
CS Div. Nephrol., Univ. Washington Med. Sch., 1959 N.E. Pacific St., Box 356521, Seattle, WA 98195, USA
SO Journal of the American Society of Nephrology, (June, 1998) Vol. 9, No. 6, pp. 986-993. print.
CODEN: JASNEU. ISSN: 1046-6673.
DT Article
LA English
ED Entered STN: 22 Jul 1998
Last Updated on STN: 22 Jul 1998

L4 ANSWER 11 OF 12 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 1997:503330 BIOSIS
DN PREV199799802533
TI Involvement of p21-WAF1/Cip1, CDK4 and Rb in activin A mediated signaling leading to hepatoma cell growth inhibition.
AU Zauberman, Ayelet; Oren, Moshe; Zipori, Dov [Reprint author]
CS Dep. Molecular Cell Biology, Weizmann Inst. Science, Rehovot 76100, Israel
SO Oncogene, (1997) Vol. 15, No. 14, pp. 1705-1711.
CODEN: ONCNES. ISSN: 0950-9232.
DT Article
LA English
ED Entered STN: 21 Nov 1997
Last Updated on STN: 21 Nov 1997

L4 ANSWER 12 OF 12 MEDLINE on STN DUPLICATE 2
AN 1995012824 MEDLINE
DN PubMed ID: 7927877
TI Differentiation ability and oncogenic potential of HPV-33- and HPV-33 + ras-transfected keratinocytes.
AU Gilles C; Piette J; Peter W; Fusenig N E; Foidart J M
CS Laboratory of General Biology, University of Liege, Belgium.
SO International journal of cancer. Journal international du cancer, (1994 Sep 15) Vol. 58, No. 6, pp. 847-54.
Journal code: 0042124. ISSN: 0020-7136.
CY United States
DT (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 199410
ED Entered STN: 22 Dec 1994
Last Updated on STN: 22 Dec 1994
Entered Medline: 25 Oct 1994

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=> d l4 1-12 bib ab

L4 ANSWER 1 OF 12 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 2009:136475 BIOSIS
DN PREV200900136475
TI Dietary Carbohydrate Source Alters Gene Expression Profile of Intestinal Epithelium in Mice.
AU Wang, Bing [Reprint Author]; Bobe, Gerd; LaPres, John J.; Bourquin, Leslie D.
CS Michigan State Univ, Dept Food Sci and Human Nutr, E Lansing, MI 48824 USA bourqui1@msu.edu
SO Nutrition and Cancer, (2009) Vol. 61, No. 1, pp. 146-155.
CODEN: NUCADQ. ISSN: 0163-5581.
DT Article
LA English
ED Entered STN: 18 Feb 2009
Last Updated on STN: 18 Feb 2009
AB High-sucrose consumption is associated with increased risk of human colon cancer. Our previous research indicated that high-sucrose diets (vs. cornstarch) promote intestinal epithelial cell proliferation and tumorigenesis as well as increase serum glucose and hepatic IGF-I mRNA levels in APCMin mice. To examine the role of functional pathways, in particular of IGF-I signaling, in sucrose-induced intestinal epithelial cell proliferation and tumorigenesis, we examined the effects of dietary carbohydrate source (sucrose vs. cornstarch) on gene expression in the intestinal epithelium using cDNA microarray and quantitative RT-PCR analysis. Dietary carbohydrate source significantly (P 0.05) altered mRNA expression of 109 known genes in the small intestinal epithelium, including many involved in metabolic pathways. Consumption of high-sucrose diets altered expression levels of genes involved in cell adhesion, cell cycle control, and transduction signaling, consistent with increased risk of intestinal tumorigenesis. High-sucrose intake also affected expression of genes involved in IGF-I signaling, including upregulating IGF-II and downregulating IGFBP3, which supports our hypothesis that IGF-I signaling could play a role in intestinal epithelial cell proliferation and tumorigenesis promoted by high-sucrose consumption.

L4 ANSWER 2 OF 12 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
AN 2009176133 EMBASE
TI Advances in the development of kinase inhibitor therapeutics for alzheimer's disease.
AU Savage, Mary J.
CS Merck and Company, West Point, PA 19486. mary_savage@merck.com
AU Gingrich, Diane E.
CS Cephalon, Inc., West Chester, PA 19380.
AU Savage, M. J., Dr. (correspondence)
CS Merck and Company, West Point, PA 19486. mary_savage@merck.com
SO Drug Development Research, (March 2009) Vol. 70, No. 2, pp. 125-144.
Refs: 221
ISSN: 0272-4391; E-ISSN: 1098-2299 CODEN: DDREDK
PB Wiley-Liss Inc., 111 River Street, Hoboken, NJ 07030-5774, United States.
CY United States
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LA English
SL English
ED Entered STN: 5 May 2009
Last Updated on STN: 5 May 2009
AB Pharmaceutical approaches to slow the progression of Alzheimer's disease

(AD) have focused primarily on reducing production or increasing clearance of amyloid β peptide (Aβ). Recent clinical trial results question the efficacy of targeting Aβ for treatment of mild to moderate AD, highlighting the need for alternate approaches. With the marketing of eight kinase inhibitors for oncology indications as of 2008 (Gleevec®, Tarceva®, Nexavar®, Sutent®, Rapamune®, Sprycel®, Tasigna®, and Tykerb®) and current clinical trials of more than 150 others for a number of indications, the progress that has been made in improving the selectivity and pharmaceutical properties of this class of compounds suggests that targeting neurodegenerative diseases such as AD may be possible. The present review describes a number of kinase targets for AD that have been studied in relation to tau protein pathology, neuroinflammation and neuron loss, in addition to amyloid pathology. .COPYRGT. 2009 Wiley-Liss, Inc.

L4 ANSWER 3 OF 12 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 2009:277956 BIOSIS
DN PREV200900277956
TI beta-tting on p63 as a Metastatic Suppressor.
AU Clohessy, John G.; Pandolfi, Pier Paolo [Reprint Author]
CS Harvard Univ, Sch Med, Beth Israel Deaconess Canc Ctr, Canc Gene Program, Boston, MA 02215 USA
ppandolf@bidmc.harvard.edu
SO Cell, (APR 2 2009) Vol. 137, No. 1, pp. 28-31.
CODEN: CELLB5. ISSN: 0092-8674.
DT Article
Editorial
LA English
ED Entered STN: 30 Apr 2009
Last Updated on STN: 30 Apr 2009
AB Although much is known about the genes that promote metastasis, few suppressors of metastasis have been found. Adorno et al. (2009) now identify p63 as a potent suppressor of metastasis and uncover an intricate mechanism for the inactivation of metastasis in cancer cells in response to transforming growth factor beta.

L4 ANSWER 4 OF 12 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 2006:263861 BIOSIS
DN PREV200600263250
TI Hematopoietic stem cell exhaustion impacted by p18(INK4C) and p21(Cip1/Waf1) in opposite manners.
AU Yu, Hui; Yuan, Youzhong; Shen, Hongmei; Cheng, Tao [Reprint Author]
CS Hillman Canc Ctr Res Pavil, 5117 Ctr Ave,Room 2-42E, Pittsburgh, PA 15213 USA
chengt@upmc.edu
SO Blood, (FEB 1 2006) Vol. 107, No. 3, pp. 1200-1206.
CODEN: BLOOAW. ISSN: 0006-4971.
DT Article
LA English
ED Entered STN: 10 May 2006
Last Updated on STN: 10 May 2006
AB Transplantation-associated stress can compromise the hematopoletic potential of hematopoietic stem cells (HSCs). As a consequence, HSCs may undergo "exhaustion" in serial transplant recipients, for which the cellular and molecular bases are not well understood. Hematopoietic exhaustion appears to be accelerated in the absence of p21(Cip1/Waf1) (p21), a cyclin-dependent kinase inhibitor (CKI) in irradiated hosts. Our recent study demonstrated that unlike loss of p21, deletion of p18(INK4C) (p18), a distinct CKI, results in improved long-term engraftment, largely because of increased self-renewing divisions of HSCs in vivo. We show here that HSCs deficient in p18 sustained their competitiveness to

wild-type HSCs from unmanipulated young mice, and retained multilineage differentiation potential after multiple rounds of serial bone marrow transfer over a period of more than 3 years. Further, p18 absence significantly decelerated hematopoietic exhaustion caused by p21 deficiency. Such an effect was shown to occur at the stem cell level, likely by a counteracting mechanism against the cellular senescence outcome. Our current study provides new insights into the distinct impacts of these cell-cycle regulators on HSC exhaustion and possibly HSC aging as well under proliferative stress, thereby offering potential pharmacologic targets for sustaining the durability of stressed HSCs in transplantation or elderly patients.

L4 ANSWER 5 OF 12 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 2007:260865 BIOSIS
DN PREV200700270932
TI Cultured human mammary epithelial cell senescence barriers and hTERT expression.
AU Stampfer, Martha R. [Reprint Author]; Tlsty, Thea; Bazarov, Alex; Yaswen, Paul; Garbe, James
CS Lawrence Berkeley Natl Lab, Berkeley, CA USA
SO Proceedings of the American Association for Cancer Research Annual Meeting, (APR 2005) Vol. 46, pp. 666-667.
Meeting Info.: 96th Annual Meeting of the American-Association-for-Cancer-Research. Anaheim, CA, USA. April 16 -20, 2005. Amer Assoc Canc Res.
ISSN: 0197-016X.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 25 Apr 2007
Last Updated on STN: 11 Jul 2007

L4 ANSWER 6 OF 12 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2004:143261 CAPLUS
DN 140:176313
TI casein kinase I gamma-1 isoforms (CSNK1G1s) as modifiers of the p21 pathway and uses thereof in diagnosis, therapy and drug screening
IN Francis-Lang, Helen; Friedman, Lori; Kidd, Thomas; Roche, Siobhan; Zhang, Haiguang
PA Exelixis, Inc., USA
SO PCT Int. Appl., 69 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 8

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| PI WO 2004015071 | A2 | 20040219 | WO 2003-US24551 | 20030806 |
| WO 2004015071 | A3 | 20040812 | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW | | | | |
| RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| CA 2494236 | A1 | 20040219 | CA 2003-2494236 | 20030806 |

AU 2003263995 A1 20040225 AU 2003-263995 20030806
EP 1534852 A2 20050601 EP 2003-784937 20030806
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
JP 2005534334 T 20051117 JP 2004-527773 20030806
US 20050251870 A1 20051110 US 2005-523588 20050204
PRAI US 2002-401739P P 20020807
WO 2003-US24551 W 20030806
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
AB The invention has designed a dominant loss of function screen to identify genes that interact with the cyclin dependent kinase inhibitor p21 in Drosophila. Casein kinase I gamma-1 isoform 3 (CSNK1G1) gene was identified as a modifier of the p21 pathway. Accordingly, vertebrate orthologs of these modifiers, and preferably the human orthologs, casein kinase I gamma-1 isoform (CSNK1G1) genes are attractive drag targets for the treatment of pathologies associated with a defective p21 signaling pathway, such as cancer. The invention also provides methods for utilizing these p21 modifier genes and polypeptides to identify candidate therapeutic agents that can be used in the treatment of disorders associated with defective p21 function.
OSC.G 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

L4 ANSWER 7 OF 12 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 2002:112722 BIOSIS
DN PREV200200112722
TI Differential cyclin-dependent kinase inhibitor CKI and INK4A expression in canine breast cancer.
AU Lynn, Kristie A. [Reprint author]; DeInnocentes, Patricia [Reprint author]; Gwin, William R. [Reprint author]; Bird, R. Curtis [Reprint author]
CS Pathobiology, Auburn University, Auburn, AL, USA
SO Molecular Biology of the Cell, (Nov, 2001) Vol. 12, No. Supplement, pp. 11a. print.
Meeting Info.: 41st Annual Meeting of the American Society for Cell Biology. Washington DC, USA. December 08-12, 2001. American Society for Cell Biology.
CODEN: MBCEEV. ISSN: 1059-1524.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 30 Jan 2002
Last Updated on STN: 26 Feb 2002

L4 ANSWER 8 OF 12 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN DUPLICATE 1
AN 1999268046 EMBASE
TI Angiotensin II stimulates serine phosphorylation of the adaptor protein Nck: Physical association with the serine/threonine kinases Pak1 and casein kinase I.
AU Voisin, Laure; Meloche, Sylvain (correspondence)
CS Centre de Recherche, Ctr. Hosp. de l'Univ. de Montreal, University of Montreal, 3850 St. Urbain, Montreal, Que. H2W 1T8, Canada. meloches@ere.umontreal.ca
AU Larose, Louise
CS Department of Experimental Medicine, McGill University, Montreal, Que. H3A 2B2, Canada.
AU Meloche, Sylvain (correspondence)
CS Centre de Recherche, Centre hospitalier Univ. de Montreal, Campus Hotel-Dieu, 3850 St. Urbain, Montreal, Que. H2W 1T8, Canada. meloches@ere.umontreal.ca

SO Biochemical Journal, (1 Jul 1999) Vol. 341, No. 1, pp. 217-223.
Refs: 44
ISSN: 0264-6021 CODEN: BIJOAK
CY United Kingdom
DT Journal; Article
FS 029 Clinical and Experimental Biochemistry
LA English
SL English
ED Entered STN: 12 Aug 1999
Last Updated on STN: 12 Aug 1999
AB Nck is a small adaptor protein consisting exclusively of three SH3 domains and one SH2 domain. Nck is thought to have an important role in cell signalling by coupling receptor tyrosine kinases, via its SH2 domain, to downstream SH3-binding effectors. We report here that angiotensin II, working through the AT1 receptor subtype, stimulates the phosphorylation of Nck in rat aortic smooth muscle cells. Phosphopeptide mapping analysis revealed that Nck is phosphorylated on four peptides containing exclusively phosphoserine in quiescent cells. Treatment with angiotensin II resulted in increased phosphorylation of these four peptides, without the appearance of new phosphopeptides. We show that Nck, via its SH3 domains, specifically binds three major phosphoproteins of 95, 82 and 66 kDa both in vitro and in intact cells. Notably, the phosphorylation of these Nck-binding proteins was found to increase in parallel with that of Nck on stimulation by angiotensin II. One candidate for the 66 kDa phosphoprotein is the serine/threonine kinase p21-activated kinase 1 (Pak1), which was found to form a stable complex with Nck in aortic smooth muscle cells. We have also identified the γ2 isoform of casein kinase I as another protein kinase that associates with Nck in these cells. These findings indicate that Nck is a target of G-protein-coupled receptors and suggest a role for Pak1 and casein kinase I-.gamma.2 in downstream signalling or regulation of the AT1 receptor.

L4 ANSWER 9 OF 12 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 1999:10466 BIOSIS
DN PREV199900010466
TI Lovastatin mediated G1 arrest in normal and tumor breast cells is through inhibition of CDK2 activity and redistribution of p21 and p27, independent of p53.
AU Rao, Sharmila; Lowe, Michael; Herliczek, Thaddeus W.; Keyomarsi, Khandan [Reprint author]
CS Lab. Diagnostic Oncol., Div. Molecular Med., Wadsworth Cent., Albany, NY 12201-0509, USA
SO Oncogene, (Nov. 5, 1998) Vol. 17, No. 18, pp. 2393-2402. print.
CODEN: ONCNES. ISSN: 0950-9232.
DT Article
LA English
ED Entered STN: 11 Jan 1999
Last Updated on STN: 11 Jan 1999
AB Previously, we reported that lovastatin, a potent inhibitor of the enzyme HMG CoA reductase also acts as an antimitogenic agent by arresting cells in the G1 phase of the cell cycle resulting in cell cycle-independent alteration of cyclin dependent kinase inhibitors (CKIs). In the present study we have investigated the nature of the CKIs (p21 and p27) alterations resulting in G1 arrest in both normal and tumor breast cell lines by lovastatin. We show that even though lovastatin treatment causes G1 arrest in a wide variety of normal and tumor breast cells irrespective of their p53 or pRb status, the p21 and p27 protein levels are not increased in all cell lines treated suggesting that the increase in p21 and p27 protein expression per se is not necessary for lovastatin mediated G1 arrest. However, the binding of p21 and

p27 to CDK2 increases significantly following treatment of cells with lovastatin leading to inhibition of CDK2 activity and a subsequent arrest of cells in G1. The increased CK1 binding to CDK2 is achieved by the redistribution of both p21 and p27 from CDK4 to CDK2 complexes subsequent to decreases in CDK4 and cyclin D3 expression following lovastatin treatment. Lastly, we show that lovastatin treatment of 76N-E6 breast cell line with an altered p53 pathway also results in G1 arrest and similar redistribution of CKIs from CDK4 to CDK2 as observed in other breast cell lines examined. These observations suggest that lovastatin induced G1 arrest of breast cell lines is through a p53 independent pathway and is mediated by decreased CDK2 activity through redistribution of CKIs from CDK4 to CDK2.

L4 ANSWER 10 OF 12 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 1998:326689 BIOSIS
DN PREV199800326689
TI The cyclin kinase inhibitor p21WAF1,CIP1 is increased in experimental diabetic nephropathy: Potential role in glomerular hypertrophy.
AU Kuan, Chia-Jen; Al-Douahji, Mouhannad; Shankland, Stuart J. [Reprint author]
CS Div. Nephrol., Univ. Washington Med. Sch., 1959 N.E. Pacific St., Box 356521, Seattle, WA 98195, USA
SO Journal of the American Society of Nephrology, (June, 1998) Vol. 9, No. 6, pp. 986-993. print.
CODEN: JASNEU. ISSN: 1046-6673.
DT Article
LA English
ED Entered STN: 22 Jul 1998
Last Updated on STN: 22 Jul 1998
AB High glucose inhibits mesangial cell proliferation in vitro and induces hypertrophy in mesangial cells in culture and in experimental diabetic nephropathy. Cell growth is ultimately controlled at the level of the cell cycle by cell cycle regulatory proteins. Cell cycle progression requires that cyclin-dependent kinases be activated by cyclins. Cyclin kinase inhibitors (CKI) inactivate cyclin-dependent kinases, causing cell cycle arrest. In the current study, high glucose-induced mesangial cell hypertrophy in vitro is shown to be associated with increased levels of the CK1 p21, but not p27. In the streptozotocin model of experimental diabetes in the mouse, glomerular hypertrophy was associated with a selective increase in p21 expression, whereas the levels of the CKI p27 and p57 did not chance. Unlike many other forms of glomerular injury, diabetic nephropathy was not associated with increased apoptosis. These results support a role for p21 in causing glomerular cell hypertrophy in diabetic nephropathy.

L4 ANSWER 11 OF 12 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 1997:503330 BIOSIS
DN PREV199799802533
TI Involvement of p21-WAF1/Cip1, CDK4 and Rb in activin A mediated signaling leading to hepatoma cell growth inhibition.
AU Zauberman, Ayelet; Oren, Moshe; Zipori, Dov [Reprint author]
CS Dep. Molecular Cell Biology, Weizmann Inst. Science, Rehovot 76100, Israel
SO Oncogene, (1997) Vol. 15, No. 14, pp. 1705-1711.
CODEN: ONCNES. ISSN: 0950-9232.
DT Article
LA English
ED Entered STN: 21 Nov 1997
Last Updated on STN: 21 Nov 1997
AB Cytokines are growth inhibitory in a target cell specific manner. The

signaling pathways that characterize each cell type play a crucial role in determining the responsiveness to cytokine triggering. Activin A has been shown to suppress the growth of primary hepatocytes. Similarly, the human HepG2 hepatoma cell line was growth arrested by activin A as judged by lack of cell proliferation and suppression of DNA synthesis. In HepG2 cells activin A further induced accumulation of retinoblastoma protein in the hypophosphorylated form known to prevent entrance into S phase. This finding implies the involvement of cyclin dependent kinases and CDK inhibitors. Examination of HepG2 cells following addition of activin A revealed reduced expression of CDK4 and conversely, an increase in the CK1 p21-WAF1/Cip1. This
accumulation of p21-WAF1/Cip1 protein was
partly due to increased transcriptional activity. Functional inactivation of p53, using a miniprotein that oligomerizes with p53 and abrogates DNA binding, abolished the ability of activin A to induce transcriptional activation from the p21-WAF1/Cip1 promoter.
Thus, activin A, like transforming growth factor beta, seems to suppress cell growth through the downstream target Rb. However, each of these cytokines seem to operate through a distinct pathway.

L4 ANSWER 12 OF 12 MEDLINE on STN DUPLICATE 2
AN 1995012824 MEDLINE
DN PubMed ID: 7927877
TI Differentiation ability and oncogenic potential of HPV-33- and HPV-33 + ras-transfected keratinocytes.
AU Gilles C; Piette J; Peter W; Fusenig N E; Foidart J M
CS Laboratory of General Biology, University of Liege, Belgium.
SO International journal of cancer. Journal international du cancer, (1994 Sep 15) Vol. 58, No. 6, pp. 847-54.
Journal code: 0042124. ISSN: 0020-7136.
CY United States
DT (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 199410
ED Entered STN: 22 Dec 1994
Last Updated on STN: 22 Dec 1994
Entered Medline: 25 Oct 1994
AB Five HPV-33-immortalized and 5 HPV-33 + ras-transfected cell lines were characterized in terms of growth in soft agar, tumorigenic potential in nude mice, p21 expression, morphology and expression of differentiation markers in organotypic cultures. No striking differences were observed between the HPV-33-immortalized cell lines and their corresponding ras-transfected counterparts as regards their tumorigenicity in nude mice (only one cell line was able to develop tumors in nude mice) or their behavior on lifted collagen gels. However, all the ras-transfected cell lines gave rise to colonies in soft agar while only 2 HPV-33-transfected lines (CK1 and CK4) displayed this property. The 10 cell lines could be divided into 2 groups with respect to their phenotype in monolayer and in organotypic cultures. Lines from group I (CK2, 3, 5 and their ras-transfected homologous lines) shared a typical epithelial phenotype in monolayer and the ability (a) to form an epithelium similar to a CIN-III lesion and (b) to strongly express keratins K1-K10 and involucrin in organotypic cultures. On the other hand, for the lines from group II (CK1, CK4, CK1EJ7 and CK4EJ5), there was a correlation between an elongated phenotype in monolayer and the property (a) to form a structure similar to a microinvasive carcinoma and (b) to express vimentin and keratins K8-K18. These cell lines, exhibiting various transformation-associated alterations, can be

considered as an in vitro model representing various stages of HPV-33-associated cervical carcinogenesis.

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